MELLANBY, EDWARD (London). **The experimental production of deafness in young animals by diet.**

Young dogs, 6—8 weeks old, were fed on a diet, complete so far

$T_1(a)$

It is postulated that ingested fat is not immediately incorporated into the carcass fat since a greater ketonuria occurs during fasting after its administration than in control animals.

[1] Deuel, Harry J. Jr., Hallman, Lois F., and Murray, Sheila, J. Biol. Chem. **119,** 257 (1937).

as was known, except that it was deficient in vitamin *A* and carotene. The dietetic ingredients consisted of separated milk, lean meat, olive or peanut oil, yeast, lemon or orange juice, sodium chloride and irradiated ergosterol, together with cereals such as white flour or oatmeal. The cereal formed a large part of the diet, namely, about 60% of the total bulk.

It was found that after about 5 months of this diet, although the general health of the animals was fairly good, they became deaf, in some cases completely. The rate of onset of the deafness varied from animal to animal, according to the stores of vitamin *A* in the body at the beginning of the experiment and the degree of deficiency of this substance. As far as present experience goes, complete destruction of the cochlear division of the 8th nerve ultimately takes place. The vestibular division suffers likewise but not so severely as the cochlear division. The destruction of the vestibular division is accompanied by loss of sense of position of the head and great incoordination of movement of the animal.

Degenerative changes in both parts of the 8th nerve and in many other peripheral and central nerves produced by this dietetic defect have previously been described by the author in papers published from 1926 onwards. It had not, however, been previously appreciated, until sections were made of the cochlea itself, how complete may be the destruction of both branches of the 8th nerve.

Apart from the destruction of the central nerve fibres, the spiral ganglion often completely disappears together with the peripheral branches of this ganglion to the organ of *Corti*. In course of time destructive changes in the peripheral sensory mechanisms also occur. Many of the cells of the organ of *Corti* may, for instance, disappear, while the epithelium of the ampullae in the semi-circular canals may lose its hairs and show further degenerative changes.

Accompanying the destruction of these nerves and possibly responsible for it, are changes in the bones of the labyrinthine capsule itself. Great overgrowth of bone can be seen in the modiolus and in the periosteal bone of the capsule. The bony overgrowth in the modiolus causes compression of the nerves, while the overgrowth of the periosteal bone of the labyrinthine capsule causes lengthening of the internal auditory meatus and so a stretching of the nerve between its origins in the spiral and Scarpa ganglia until it enters the brain. These mechanical effects of pressure and stretching can be inferred from the histological preparations of the cochlea and labyrinthine bone. Macroscopic examination of the skull also reveals that the bone overgrowth is not confined to the labyrinthine capsule. Other bones are hypertrophied in animals brought up on these diets and there is evidence of pressure being brought to bear upon other cranial nerves and also on the arteries entering the skull by bony occlusion. To what extent pressure and stretching caused by bone overgrowth is responsible for other widespread nerve degeneration previously reported in these animals is not yet clear.

The addition of a source of vitamin *A* or carotene to the above diets prevents all these changes. Also, if the cereal portion of the diet is replaced by potato, even without the addition of extra vitamin *A* or carotene, the bones and nerves of the labyrinthine capsule are much less affected.

Whether these changes in dogs above described have any bearing on the problem of deafness in human beings cannot be stated. It is clear, however, that both the bone overgrowth and the nerve degeneration are much more easily produced in young animals than in adults by these nutritional defects.

Emerson, O. H. (Berkeley). **The structure of beta and gamma tocopherols.**

On oxidizing beta and gamma tocopherol with chromic acid, the same $C_{21}H_{40}O_2$ lactone was obtained that had previously been obtained by *Fernholz* by the oxidation of α-tocopherol. Pyrolysis of gamma-tocopherol yielded pseudo cumo hydroquinone. Hence, beta and gamma tocopherol differ from alpha only in the absence of one of the methyl groups attached to the aromatic ring.

Mattill, H. A. (Iowa City). **Vitamin E and nutritional muscular dystrophy in rabbits.**

Several investigators have stated that the muscular degeneration first demonstrated by *Goettsch* and *Pappenheimer*[1]) in young rabbits on rations containing lard and especially cod liver oil is not preventable by including various vegetable oils in the diet. Even wheat germ oil, which is rich in Vitamin E, is by itself not adequate to prevent the disease, according to *Morgulis* and *Spencer*[2]); another factor, found in wheat germ, lettuce and alfalfa, must also be present for prevention or cure. The recent observation of *McCay*, *Paul* and *Maynard*[3]), that hydrogenated cod liver oil in a natural grain diet produces no ill effects suggests that auto-oxidation of the unsaturated fatty acids may, indirectly, be the principal cause of the disorder. Vitamin E is known to be destroyed quickly in the presence of readily auto-oxidizable animal fats.

When an efficient antioxidant is included in a synthetic diet containing no sources of Vitamin E, animals succomb as rapidly as on the basal diet alone. The presence of 2% of wheat germ oil in the basal diet has, in our experience, been adequate to prevent the disease for many months, during which the animals grew normally. Thus far,